

Poison Oak Desensitization

THE SKIN ERUPTION produced by the *Rhus* plants—poison oak, poison ivy and poison sumac—is an allergic contact dermatitis occurring in patients sensitized to the plant oleoresin. Highly sensitive patients may react to casual or inapparent contact, such as from burning leaves or by secondary transmission from household pets or horses. Poison oak plants are widely distributed throughout the West and are difficult to eradicate. The disease is a serious problem for allergic persons working outdoors—such as foresters, rangers and telephone linemen.

Desensitization by oral or parenteral administration of *Rhus* extracts has been used for many years, but the value of this form of prophylaxis has never been established fully. Individual reports of successful desensitization are difficult to accept, because natural loss of sensitivity through repeated contact ("hardening") or simple avoidance of contact with the plant could account for some of the apparent successes. Commercially-available extracts are sometimes inactive, and active extracts may induce serious reactions in highly sensitive persons.

Epstein and co-workers recently conducted a study of the effectiveness of desensitization using the purified allergen urushiol extracted from poison oak leaves collected in California. The compound was administered orally to volunteers with high-grade sensitivity, and the effectiveness was determined by repeated titration-endpoint patch testing, using a double blind experimental design. The results were not dramatic, but in some patients there was significant reduction in sensitivity, which was dose-related. Some of the placebo-treated subjects showed decreased sensitivity, probably attributable to the repeated patch testing. Pruritus and skin rashes were observed in some instances. Attempts to inject urushiol intramuscularly were abandoned because of unacceptable inflammation at the injection sites.

The results of this study should encourage other investigators to pursue the search for a more effective and less toxic desensitizing procedure for this distressing condition. However, there is no evidence at present that currently available *Rhus* extracts can be recommended for desensitization.

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REFERENCE

Epstein WI, Baer H, Dawson CR, et al: Poison oak hyposensitization: Evaluation of purified urushiol. *Arch Dermatol* 109:356-360, Mar 1974

Viral Infections as Triggers of Asthmatic Attacks

MOST ASTHMATIC PATIENTS have attacks of asthma in association with respiratory infections. This phenomenon occurs in persons with intrinsic asthma, as well as in those patients with atopic allergy. Cultures of the respiratory secretions during such attacks generally fail to show specific pathogenic bacteria. There is a strong suggestion on clinical grounds that the respiratory infections most likely to exacerbate asthma are viral in cause. Several recent studies have helped to clarify the role of specific viruses as precipitating factors in asthma.

McIntosh and co-workers conducted a two-year study with 32 asthmatic children under age 5. The children lived in a residential treatment center, and most of them were atopic. It was found that 42 percent of their asthmatic attacks were associated with viral respiratory infections verified by cultures, in contrast to a lack of correlation with bacteria in their secretions. Respiratory syncytial virus infections almost always precipitated an attack of asthma, which was usually accompanied by bronchopneumonic infiltrates. Parainfluenza and coronavirus infections occurred frequently but less consistently exacerbated asthma, whereas adenovirus infections usually did not cause asthma unless the infection was especially severe. A single mild epidemic of influenza A₂/Hong Kong caused no wheezing in this population.

In a study by Minor and associates, older children and adults who were outpatients were studied. These patients were mostly nonatopic and were carefully selected for previous history of infection-provoked asthma. Rhinovirus infections usually triggered asthma when the infection was symptomatic, almost invariably when symptoms were severe. Influenza A and respiratory syncytial virus infections also provoked wheezing when infections were symptomatic, but adenovirus infections did not. Bacterial infections failed to trigger asthmatic attacks. This study showed further that primary viral respiratory infections did not produce a secondary bacterial component.

Viral respiratory infections are clearly capable of triggering attacks of asthma in patients of all ages, and they do so quite frequently. At least several viruses are asthmagenic, and the severity of the infection may be more important than the specific virus.

The mechanism is unknown, but viral-induced